Isotropic continuum damage/repair model for alveolar bone remodeling

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Abstract

Several authors have proposed mechanical models to predict long term tooth movement considering both the tooth and its surrounding bone tissue as isotropic linear elastic materials coupled to either an adaptative elasticity behavior or an update of the elasticity constants with density evolution. However, tooth movements obtained through orthodontic appliances result from a complex biochemical process of bone structure and density adaptation to its mechanical environment, called bone remodeling. This process is far from linear reversible elasticity. It leads to permanent deformations due to biochemical actions. The proposed biomechanical constitutive law, inspired from [1], is based on a elasto-viscoplastic material coupled with Continuum isotropic Damage Mechanics ([1] considered only the case of a linear elastic material coupled with damage). The considered damage variable is not actual damage of the tissue but a measure of bone density. The damage evolution law therefore implies a density evolution. It is here formulated as to be used explicitly for alveolar bone, whose remodeling cells are considered to be triggered by the pressure state applied to the bone matrix. A 2D model of a tooth submitted to a tipping movement is presented. Results show a reliable qualitative prediction of bone density variation around a tooth submitted to orthodontic forces.

Key words: biomechanics, bone remodeling, orthodontics, damage/repair model, continuum damage mechanics, elastoplasticity

1. Introduction

One of the guiding principles in orthodontics is to gradually impose progressive and irreversible bone deformations. By optimizing load positions and intensities, treatment can be reduced both in time and cost. This optimization requires a mechanical model of the biochemical phenomena involved and the activated dental movement. The goal of this work is to provide a constitutive model able to simulate those coupled phenomena.

Dental movement is achieved through a biochemical process of skeletal adaptation to mechanical stimuli called bone remodeling, controlled by bone cells and first described by Wolff in 1892 [2] (as cited in [3] among others). Therapeutic forces applied through orthodontic appliances change the physiological equilibrium. Loading of the skeletal system is altered and bone

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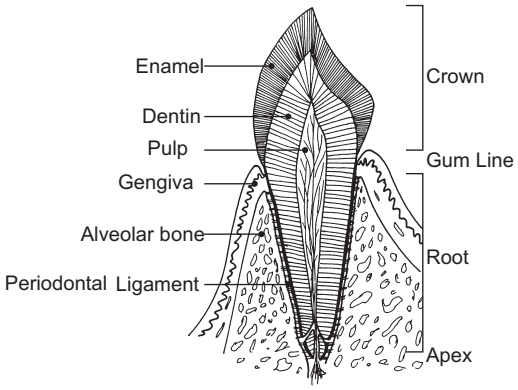


Figure 1: Tooth and surrounding tissue - anatomy

remodeling cells are triggered to modify the bone shape and density in order to achieve a new equilibrium and adjust the stress level. This state will be maintained until new mechanical external conditions trigger new remodeling events. Beyond this remodeling process due to a change of external conditions, a physiological remodeling process also exist. Its function is to maintain bone cells in use and to ensure a relatively high turn-over of the tissue. To distinguish between these two processes, the first one is often referred as bone modeling or external remodeling and the second as bone (internal) remodeling. However, within the orthodontics literature, the designation “remodeling” is used for both processes ; one would read [4] for a discussion about the paradigm it causes. The word “bone remodeling” will be here used for the process due to external mechanical events. Internal remodeling is not modeled in this work. We consider, as a simplifying assumption, that its function is to renew the cells with no alteration of the overall bone mechanical properties.

For most types of bones, remodeling processes take place in order to adjust the amount of tissue and its topology according to long term loading conditions, following what is called “Wolff’s law” of bone adaptation [5, 6, 7]. Bone resorption occurs when disuse is observed. This resorption tends to decrease the amount of bone where it is of no mechanical relevance. Bone apposition occurs in overloaded conditions, in order to reinforce bone where it is necessary. The bone therefore adapts its density in such a way to achieve an homeostatic state of stresses. Besides the density change, remodeling also occurs to change the bone topology, mainly in trabecular tissue for which the trabeculae tend to align along the principal stress directions. Bone remodeling therefore depends not only on the stresses intensity but also on their direction.

Contrary to the majority of bones, alveolar bone remodeling seems on a macroscopic scale to depend mainly on the pressure state [4, 8, 9]. One can indeed observe apposition on the tension side of a tooth when loaded with an abnormal mechanical environment, such as the one obtained with orthodontics appliances, as well as resorption on the compression side. The actual biomechanical processes causing such a difference is not quite clear and uniformly accepted among biology and biochemistry literature (see discussions in [7, 10, 11]). To model these processes, several authors do not consider non linearities of the bone. Some focus only on the initial tooth mobility and extrapolate their results with an adaptative elasticity framework as described in [3] or a density update [4, 12] or another built-in remodeling law applied as a second step to the finite element computation [8, 13, 14]. Others focus on the periodontal

ligament (PdL) non linear response [15, 16, 17, 18, 19]. Its non linearity and different behaviors in traction and compression leads to opposite loading conditions of the bone on each side of the tooth. The compressive side of the tooth would cause underuse of the bone and therefore resorption while the traction side would get overuse and apposition. However, when no non-linearities are considered in the PdL, no difference in the stress level on both side of a tooth can be observed. A non pressure dependent remodeling law used for the alveolar bone would therefore lead either to apposition or to resorption on both sides.

Instead of focusing on the PdL response, the present work concentrates on the bone behavior during remodeling. We assume the pressure state (positive or negative) of the bone matrix as the key stimulus to differentiate apposition and resorption in overloaded conditions. This can be justified as follows. The cell supply needed for bone remodeling is performed by the vasculature, however, the alveolar bone main cell supply is done through the periodontal ligament. As this membrane's stiffness is much less than the surrounding tissue's, the strain level is high. If the hydrostatic stress level is higher than the blood vessels'internal pressure, the blood flow is stopped as well as the remodeling cell supply to the bone. The remodeling process is therefore triggered by the PdL pressure and stopped when these stresses are too high. We therefore assume that it is the same stimulus which is responsible for the differentiation between apposition and resorption as well as for the triggering of the phenomenon.

Within the diverse approaches that have been adopted to model bone remodeling processes, most of them are qualified as phenomenological models. These are models that do not try to predict the evolution of the microstructure and biological constitution of a tissue or an organ as a consequence of the mechanical environment (contrary to Mechanobiological models) but whose goal is to predict the mechanical behavior (movement, strains and stresses) of a tissue or an organ, taking into account the applied loads, its microstructure and the constraints imposed by other organs. Most of these models admit the existence of a certain mechanical stimulus (input) that produces bone apposition or resorption (output) in such a way that the stimulus tends to a given physiological level in the long-term (homeostasis). Among these phenomenological models, the definition of a remodeling stimulus uses a wide range of mechanical measures : stresses, strains, strain energy density, strain rate or even damage.

The original model which is proposed in this work is built on a damage/repair based model, which is therefore a phenomenological model, stated first by Doblaré and co-workers [1, 20]. This model has been chosen as a working framework because it is one of the few models whose stimulus variation is justified through thermodynamical concepts of continuum mechanics. It is here extended and enhanced in order to be used for the alveolar bone and therefore it takes into consideration the pressure state of the tissue as one of the stimuli for bone remodeling. It is also coupled to an elasto-viscoplastic material behavior in order to capture permanent strains of the tissue. The proposed model can therefore be used to represent permanent irreversible tooth displacement and alveolar bone deformation due not only to remodeling but also to permanent deformation of the bone (and therefore plasticity-like, although it is clear that the relevant inelastic process is different from that of the classical metal plasticity). It can also be used to describe a fracture process with a plasticity-like yield function to model the envelope of bone failure. The main originalities with respect to [1] are therefore, first, an extension for alveolar bone pressure dependency of the remodeling rate and, second, a finite strain framework within which the model is written, allowing for the use of a elasto-plastic model for the bone matrix.

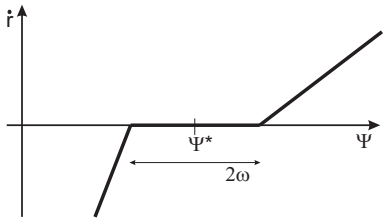


Figure 2: Stanford remodeling rate

2. Material and Methods

Based on physical experiments [21], one can show that remodeling occurs to modify the bone density proportionately to the bone matrix density, ρ_0 and as a function of a remodeling rate $\dot{r}$ $[mm/s]$,

$$\dot{\rho} = kS_v\rho_0 \ \dot{r} \tag{1}$$

The terms kS_v accounts for the available bone specific surface area (S_v, internal surface area per unit volume, related to the density, $[mm^2/mm^3]$) as defined in [22].

This remodeling rate, $\dot{r}$, is shown [21, 23, 24, 25] to be a non-linear function of the deviation from a given mechanical stimulus (ψ, a daily stimulus at tissue level function of the strain energy) from an homeostatic value ($\psi^\star$). The remodeling process thus tends to reduce this deviation. This model, known as the Stanford model, considers that the bone tissue needs a certain level of mechanical stimulus to maintain homeostasis and auto-regulates itself to maintain such a level. The remodeling rate also takes into consideration the existence of a lazy zone (width of 2ω) within which no remodeling is achieved (Fig. 2). The actual existence of this lazy zone is a concept which is not uniformly accepted among the biological community. Some people do seem to believe that this zone has been introduced by the numerical community for convergence purpose more than for actual biological reasons.

Once the density variation is computed, the bone Young's modulus is updated according to density using a classical law based on experimental observations as proposed in [26] :

$$E = B(\rho)\rho^{\beta(\rho)} \tag{2}$$

with
$$B = 2014 \ ; \beta = 2.5 \quad : \quad \text{if } \rho \leq 1.2\ g/cc \text{ and } E \text{ expressed in } MPa$$
$$B = 1763 \ ; \beta = 3.2 \quad : \quad \text{if } \rho \geq 1.2\ g/cc \text{ and } E \text{ expressed in } MPa$$

Doblaré and co-workers [1, 20] modified this model so as to formulate it within a Continuum Damage Mechanics approach but limited to small strains elasticity for the bone matrix. We here extend the use of such a family of materials model to finite strains of the bone matrix. The use of the continuum damage theory allows to define independently the internal variables such as density and mechanical properties. It is therefore an improvement of the extension of [21] which was proposed in [25]. Indeed, in [25], Jacobs and co-workers used a global optimization function to define the remodeling stimulus and therefore the internal variables were not independent. Even though the continuum damage formulation solves this difficulty, Doblaré and co-workers limited their approach in [1] to an elastic bone matrix as was done in [3]. Therefore, they are limited to low strain levels, which is not the case here.

We propose to extend the model described in [1] to a much more generalized mechanical behavior of the bone matrix, considering it as an elasto-viscoplastic material, mandatory in order to effectively predict long term tooth movement. This model is also adapted to account for an explicit pressure dependence of the remodeling rate in the alveolar bone which, as explained in the introduction, has a different behavior in overloaded traction and compression that cannot be represented by the remodeling rate used in [1].

In order to couple continuum damage and plasticity, the use of a strain equivalence approach, relating the stress level in the damaged material ($\boldsymbol{\sigma}$) with the stress in the undamaged material (effective stress, $\tilde{\boldsymbol{\sigma}}$) that leads to the same strain, is chosen [27]. This approach keeps the physical definition of damage as related to the surface density of defects. In [1], Doblaré and co-workers used the energy equivalence approach, relating the stress level in the damaged material with the stress in the undamaged material that leads to the same strain energy, therefore loosing the relation to the surface density of defects. This choice allows to couple plasticity to damage by assuming an additive decomposition of the strain rate ($\boldsymbol{D}$) in its elastic ($\boldsymbol{D}^{el}$) and non-elastic ($\boldsymbol{D}^{pl}$) parts and expressing the plastic criterion in term of effective stresses instead of stresses which cannot be done with an energy equivalence approach.

The proposed model therefore uses the following set of equations :

$$\text{damage parameter : } d \tag{3}$$

$$\text{effective stress : } \tilde{\boldsymbol{\sigma}} = \frac{\boldsymbol{\sigma}}{(1-d)} \tag{4}$$

$$\text{strain rate : } \boldsymbol{D} = \boldsymbol{D}^{el} + \boldsymbol{D}^{pl} \tag{5}$$

$$\text{constitutive law : } \overset{\triangledown}{\tilde{\boldsymbol{\sigma}}} = \mathbb{C}_0 : (\boldsymbol{D} - \boldsymbol{D}^{pl}) \tag{6}$$

$$\text{damage variation : } \dot{d} = f(d, \boldsymbol{\sigma}, \dot{r}, \rho_0) \tag{7}$$

with d a normalized scalar damage variable, $\mathbb{C}_0$ Hooke's tensor for the undamaged material and where the $\triangledown$ sign accounts for an objective time derivative.

Stress and strain rate tensors are energy conjugated in order to be adequately used in a large deformation framework. The constitutive law (Equ.6) expresses that the effective stress rate is proportional to the elastic part of the strain rate through Hooke's tensor. The viscoplastic part of the strain rate is computed according to a given plastic criterion $f < 0$, function of a scalar representation of the effective stress ($\tilde{\sigma}_{eq}$) and a boundary that this equivalent stress cannot cross (σ_{crit}, accounting for the yield stress and viscous effects, see [28] for details):

$$f = \tilde{\sigma}_{eq} - \sigma_{crit} \tag{8}$$

This set of equation is integrated in a finite element framework according to an iterative "staggered scheme" (see [29] for details on the method) :

- Stresses are first computed at constant damage value as follows : strains are updated according to the initial state of stresses, strain and damage and with updated loading conditions. An elastic predictor for stresses is therefore computed according to Hooke's law. A plastic correction is computed in an iterative scheme if necessary. This correction is calculated through the normality rule on the plastic criterion (associated plasticity) expressed in term of effective stresses. One can show that the normal to the plastic criterion expressed

in term of effective stresses is equivalent to the normal to the criterion if it was expressed in terms of stresses, and is therefore proportional to the deviatoric part of the elastic predictor ($\tilde{\mathbf{s}}^{el}$) :

$$\boldsymbol{N} = \frac{\dfrac{\partial f}{\partial \boldsymbol{\sigma}}}{\left\| \dfrac{\partial f}{\partial \boldsymbol{\sigma}} \right\|} = \frac{\dfrac{\partial f}{\partial \tilde{\boldsymbol{\sigma}}}}{\left\| \dfrac{\partial f}{\partial \tilde{\boldsymbol{\sigma}}} \right\|} = \frac{\tilde{\mathbf{s}}^{el}}{\left\| \tilde{\mathbf{s}}^{el} \right\|} \tag{9}$$

- Once convergence of stresses at constant damage is achieved, damage is updated according to the effective stresses and plastic strains and following the damage evolution law (Equ.7).
- The elastic predictor for stresses is finally reevaluated with the new damage value, with iterations up to convergence of damage.

The yield criterion used here for the bone is the Von Mises criterion. This assumes that only shear stresses are responsible for plastic strains. As both remodeling triggering and damage variation are function of the pressure value, this assumption may be too restrictive. However, as no data is available to us on bone behavior at plasticity, we will base our study on this restrictive hypothesis.

In the case of bone remodeling, as proposed in [1], damage can be understood as a measure of the void volume fraction inside the bone tissue. Equ.(1) can therefore be formulated as a damage variation function. The damage concept used is therefore purely virtual and is actually a measure of the evolving bone density. There is no actual damage in the tissue, only a variation of density. The undamaged material is the ideal situation of bone with null porosity and perfect isotropy therefore representing fully mineralized bone. Clearly, this situation can be considered isotropic on a macroscopic level. A fully resorbed bone would have a damage value of one. However, this situation cannot strictly be treated only within the continuum damage mechanics framework as the effective stress would be undetermined. The process of bone resorption corresponds to the classical damage evolution concept, since it increases the void fraction (porosity) and therefore damage (decreases the density). However, bone apposition can reduce damage and lead to bone repair, which has to be adequately considered in this extended damage theory. Damage repair can be considered here because the total energy dissipation includes biological dissipation due to metabolism on top of the mechanical dissipation which is negative for damage repair.

In analogy with plasticity, a remodeling stimulus is identified with the variable thermodynamically associated with damage. One can therefore, as in [1], establish two damage criteria, g_o and g_u, representing the domain of the remodeling stimulus for which damage is not modified (the lazy zone) both for overuse and underuse.

$$\begin{cases} g_o &= U - (1+\Omega)U^\star < 0 \\ g_u &= 1/U - 1/((1-\Omega)U^\star) < 0 \end{cases} \tag{10}$$

These criteria depend on U, as expressed in Equ.(11), function of a strain energy density as well as the density and the number of cycles considered (n) for the applied loads, and $U^\star$, as expressed in Equ.(13), a reference homeostatic value of U. They are consistent with Beaupré and Carter's approach [21] if we relate ψ to U, $\psi^\star$ to $U^\star$ and ω to Ω, the normalized width of the lazy zone.

$$U(d, \tilde{\boldsymbol{\sigma}}) = n^{1/4} \sqrt{B(d)} \rho_0^2 \rho^{-\beta/8} \sqrt{3\bar{u}(\tilde{\boldsymbol{\sigma}})} \tag{11}$$

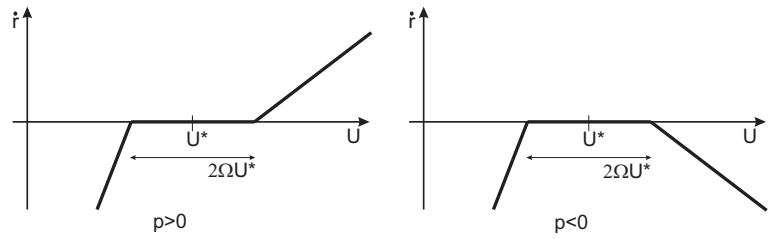


Figure 3: Pressure dependent remodeling rate, pressure p being positive in traction.

with β and B defined in Equ.(2) and $\bar{u}$, the effective elastic energy density (as also defined in [27]). Using an isotropic elastic undamaged material reduces Hooke's tensor to two parameters E and ν, Young's modulus and Poisson's ratio. The effective elastic energy density, accounting for the stress triaxiality, is therefore written as :

$$\bar{u}(\tilde{\boldsymbol{\sigma}}) = \frac{\tilde{J}_2^2}{2E}\left[\frac{2}{3}(1+\nu)+3(1-2\nu)\frac{\tilde{p}^2}{\tilde{J}_2^2}\right] \tag{12}$$

$$\text{with} \quad \tilde{J}_2 = \sqrt{\frac{3}{2}\hat{\tilde{\sigma}}_{ij}\hat{\tilde{\sigma}}_{ij}} = \frac{1}{1-d}\sqrt{\frac{3}{2}\hat{\sigma}_{ij}\hat{\sigma}_{ij}} \qquad ;\hat{\boldsymbol{\sigma}} = \mathtt{dev}(\boldsymbol{\sigma})$$

$$U^\star(d) = \psi^\star \rho^{2-5\beta/8} \tag{13}$$

Using consistency conditions and Equ.(1), one can express the damage variation as proportional to a remodeling rate such as the one proposed by the Stanford group [21, 25, 26] and presented in Fig. 2. We can therefore retrieve Equ.(14) by combining Equ.(1), Equ.(2) as well as the damage definition used in a strain equivalence approach Equ.(6).

$$\dot{d} = -\beta k S_v \dot{r}\frac{\rho_0}{\rho}(1-d) \tag{14}$$

As stated previously in the introduction of this paper, Stanford's remodeling rate cannot be applied to alveolar bone if no PdL nonlinearities are considered. In accordance with the observation of a pressure dependent phenomenon, the remodeling rate definition is modified (Fig. 3, Equ.(15)) taking into account the pressure state. This new rate is thus given by :

$$\dot{r} = \begin{cases} c_f g_o & \text{if } g_o \geq 0,\ g_u < 0 \quad \text{and} \quad p > 0 \\ -c_r g_o & \text{if } g_o \geq 0,\ g_u < 0 \quad \text{and} \quad p < 0 \\ 0 & \text{if } g_o < 0,\ g_u < 0 \\ -c_r g_u & \text{if } g_u \geq 0,\ g_o < 0 \end{cases} \tag{15}$$

where c_r and c_f are two remodeling constants respectively for bone formation and bone resorption, p is the pressure (positive in tension) and g_o and g_u are the remodeling criteria (same units as the one of stresses) respectively for bone overload and bone underload used in [1] and expressed for a strain equivalence approach in continuum damage mechanics as in Equ.(10). For numerical purpose, the differentiation between formation and resorption when $g_o \geq 0$ is not exactly at $p = 0$

but at $p = \pm\delta p \ll 1$. A linear regression of the coefficients (c_r, c_f) between $p = -\delta p$ and $p = \delta p$ is used.

As stated earlier, damage evolution is proportional to the specific surface, S_v as defined in [22]. It is written as a 5th order polynomial of the porosity and is null for null porosity as well as for full porosity ($f = 1.0$). This specific surface is introduced in order to take into account the necessity of a bone surface to exist for bone remodeling cells to act. Its presence in the damage variation law has therefore a biological justification but also serves a numerical purpose. Using this specific surface will decrease substantially the convergence problem that would arise when reaching high damage values. Indeed, the effective stress definition of the continuum damage theory in a strain equivalence approach can be written for an isotropic damage variable, d, as Equ.(6). Therefore when obtaining a damage variable equal to 1.0 (full resorption of the bone), the model cannot be used anymore. Yet, this will tend not to happen in the model because when reaching the critical value, the damage variation decreases to zero. However, the damage variation for high damage values can be high, due to the infinite limit value of the remodeling rate. It also has to be noticed that for an initial null bone porosity, there will be no damage creation, because of the zero value of the specific surface at that point. This would mean that a fully mineralized bone could not be resorbed.

Using a continuum damage approach as well as Equ.(2), one can get for both types of bone (trabecular and cortical bone), using $\rho_0 = 2.1 g/cc$ (and therefore $E_0 = 18.9 GPa$) for the fully mineralized bone :

	Density $\rho[g/cc]$		Stiffness $E[GPa]$	Damage[a] d	Porosity[b] f
Trabecular bone	1.15	$\rightarrow$	2.9	0.85	0.45
Cortical bone	1.99	$\rightarrow$	16.1	0.15	0.05

Table 1: Bone stiffness, damage and porosity as a function of density, for an isotropic material

[a] Damage is calculated from stiffness as : $d = 1 - E/E_0$.
[b] Porosity is calculated from density as : $f = 1 - \rho/\rho_0$.

For given stresses and initial density, ρ_0, damage variation with damage can be expressed for both formation and resorption stimuli, as in Fig. 4. Results will nevertheless depend on the law parameters, as n, ψ (actually only the ratio $\frac{\psi}{n^{1/4}}$ has an influence on the variation, their values have just a stretching impact) and Ω. As damage variation is positive for resorption and negative for formation when the pressure is positive, we can detect in Fig. 4 different remodeling zones as well as the lazy zone as a function of damage. As expected, damage variation for values of damage close to 1.0 tend to high (negative) values but is reduced to zero for full damage (not shown in the figure). In resorption, although the remodeling rate increases (in absolute value) for a damage decay, damage variation does not reach high values due to the tendency of the surface density to decrease faster than the remodeling rate increases. The discontinuity of damage variation for damage values of about 0.83 is due to the slight discontinuity introduced by Equ.(2) in the definition of bone Young's modulus.

The presented bone remodeling model has been implemented in Metafor [30], a home-made finite element software as a new constitutive law. Once the remodeling model has been formulated, we need to check its ability to achieve qualitative results close to the ones obtained in experimental testing of actual alveolar bone. This is accomplished in the next section in which the model is applied to study the remodeling behavior of the alveolar bone in the case of or-

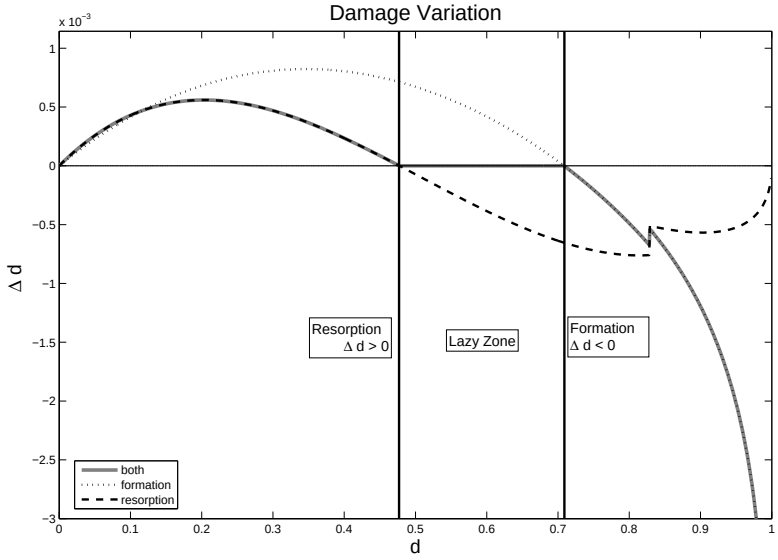


Figure 4: Damage variation as a function of damage : $n = 1000$, $\psi^\star = 10MPa$, $k = 0.5$, $\Omega = 0.25$, $p = J_2 = 0.5MPa$, $\rho_0 = 2.1g/cc$, $c_f = 5\ 10^{-5}$, $c_r = 100c_f$, the plain gray line is the actual damage variation computed (following equations Equ.(14) and Equ.(15))

thodontic treatments.

As an example, we present a 2D model of a tooth surrounded by its parodontal tissue, on the crown of which a pressure load is applied in the vestibulo-lingual direction. The aim is to predict the bone density and its evolution from an initial ideal situation (isotropic material with uniform density distribution) when loaded by forces that characterize orthodontic appliances such as brackets. As the loading applied by the use of orthodontic appliances is considered to be an homogeneously distributed pressure force on the vestibular face of the crown, it would produce a tipping movement of the tooth in the vetibulo-lingual direction. Neither this problem nor the starting situation are "real" problems, therefore the homeostatic values are not relevant. The tooth geometry is idealized, with a parabolic root surrounded by a constant thickness periodontal ligament as well as trabecular and cortical bone. The 2D discretization used here is shown in Fig. 5. The root is 12*mm* high and 6*mm* wide at the collar. The crown is 7*mm* high, the PdL's thickness is constant and of 0.2*mm*. It is surrounded by a trabecular bone of variable thickness and a cortical layer of about 0.5*mm* width. The tooth and the PdL mechanical behavior are linear elastic ($E_{\text{tooth}} \approx 20GPa$, $\nu_{\text{tooth}} = 0.3$, $E_{\text{PdL}} = 0.6MPa$, $\nu_{\text{PdL}} = 0.45$). The cortical layer as well as the trabecular bone mechanical behavior is elasto-plastic with a continuum damage model (Young's modulus as in Table 1 and Poisson's ratio of 0.3). The damage evolution follows the remodeling law proposed in this work. Finite Element analysis is performed, using finite strains code Metafor [30], considering a plane strain state as well as a quasi-static analysis. The basal bone junction is fixed in both vertical and horizontal directions. The mesh is composed of 2670 nodes and 2600 linear quadrangular elements, 60% of which is trabecular bone, 25%, tooth (crown and root) and the remaining 15% is equally distributed among PdL and cortical bone. The element sizes result in the choice of a mesh density ditribution so that element are

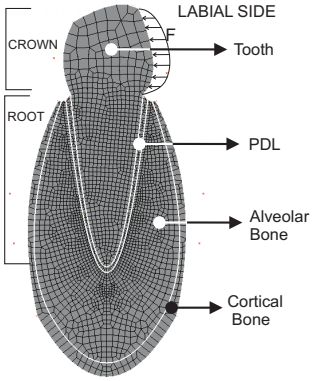


Figure 5: Tooth and surrounding tissue - mesh and geometry

smaller in the region of interest (i.e. PdL and closest surrounding tissue) than in the rest of the model, especially the crown and root as it is almost rigid compared to other tissues. Loading is performed using two levels of pressure value (corresponding to $1.0N$ and $2.0N$ force) as well as a different set of remodeling constants (see Table 2).

Force	$c_r = c_f$ $[\mu m/day]$	c_r, c_f $[\mu m/day]$
(case 1) : 1N	2.0	0.4, 4.0
(case 2) : 2N	1.0	0.2, 2.0

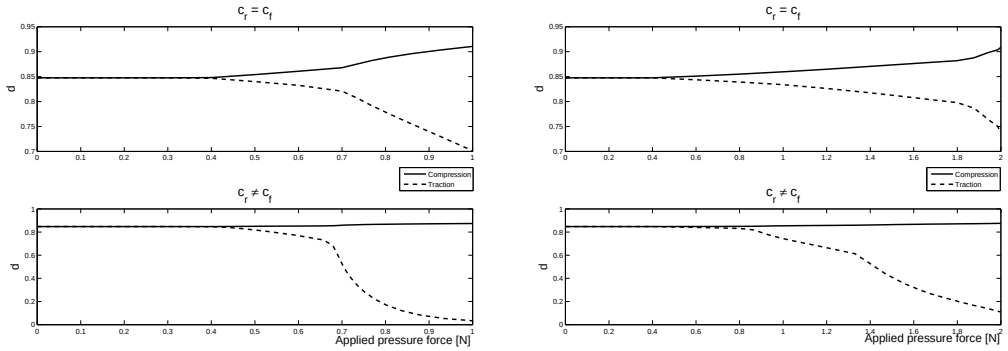
Table 2: Remodeling constants used for trabecular bone

3. Numerical results

In this section we consider the potential of the pressure dependent model to predict the density evolution of alveolar bone tissue on the case presented earlier.

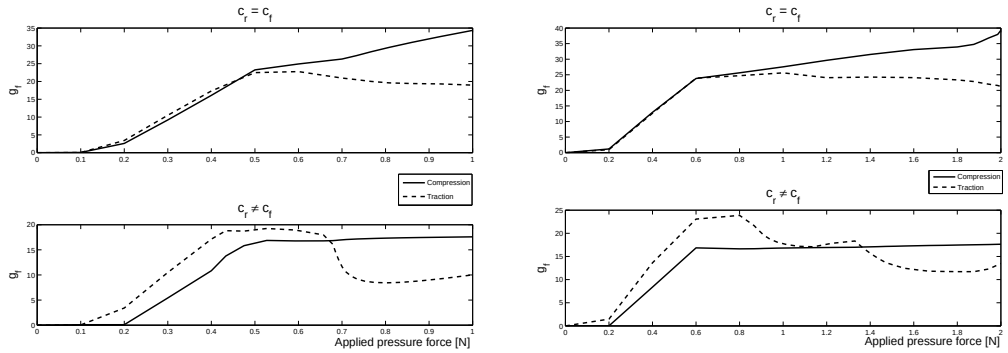
The obtained tooth movement is a rotation around a center of rotation situated at one third of the root length starting from the apex for small loads and one fifth for higher loads. This corresponds to what is exposed in the literature about tipping movement of a tooth (see [14] among others). The rotation angle is almost null for small forces, corresponding to initial tooth mobility, while it reaches about 1.5 degrees for higher forces. For the later situation, if the whole movement was a rigid rotation around the same center of rotation, one could expect a displacement of $-.1mm$ horizontally and $\pm.04mm$ vertically at the collar. However, as the bone is fixed at its base and as the rotation leads to deformation of the periodontal ligament as well as the bone, the movement actually observed leads to smaller displacements at the collar.

The initial tooth mobility depends only on the applied load and not on the remodeling model used. However the possibilities of long term tooth movement due to bone density change varies strongly with the type of model used (1 constant or 2 constants model) as well as with the homeostatic value $\psi^\star$ and the number of cycles considered. When using the same remodeling constant for both resorption and formation, one does not exactly get symmetric values for damage variation (Fig. 6, top row) because of its dependence on the damage value (Fig. 4). In the case



(a) case 1 (b) case 2

Figure 6: Damage variation at the apex for a pressure dependent model - both for the compression side for which c_r is used (plain line) and the traction one for which c_f is used (dashed line).

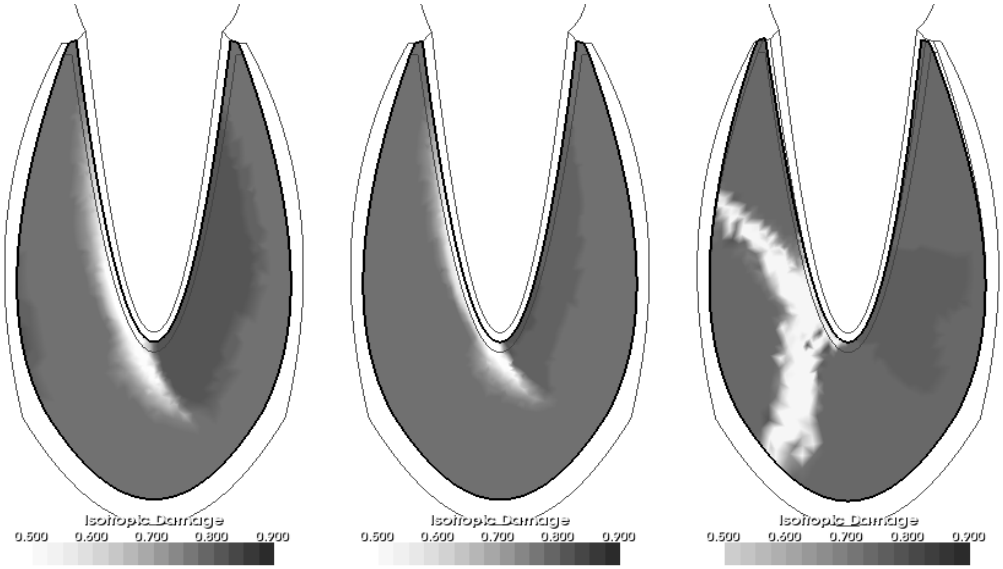


(a) case 1 (b) case 2

Figure 7: Stimulus variation at the apex for a pressure dependent model - both for the compression side for which c_r is used (plain line) and the traction one for which c_f is used (dashed line).

of two different remodeling constants (Fig. 6, bottom row), the one used in resorption, c_r, is the restrictive one because it is the one increasing the damage value. Its value is restrained so that the damage value cannot reach 1.0. The constant used in formation, c_f, can be increased almost at will (as long as numerical convergence is concerned, not on a biological point of view). However, if it is too high, the effective stiffness will increase, so will the stress. The stress state around the apex will be modified and affect the resorption side as well (with a tendency to increase the resorption).

Damage variation at the collar level is much less than at the apex or along the root for all simulations and on both sides of the root main axis. Indeed the tipping movement leads to smaller shear and hydrostatic stresses at the collar than along the root ($J_2^{\text{collar}} \approx 1MPa$, $J_2^{\text{root}} \approx 3MPa$, $p^{\text{collar}} \approx 0.2MPa$, $p^{\text{root}} \approx 1-3MPa$). It also gives a ratio hydrostatic stress to shear stress of 0.2 at the collar while it is of around 1 to 3 along the root. Therefore, the value of $\bar{u}$ is smaller at the collar than along the root (see Equ.(12)). The remodeling rate is also smaller because even tough



(a) case 1, $c_r = c_f$ (b) case 1, $c_r \neq c_f$ (c) case 2, $c_r \neq c_f$

Figure 8: Alveolar bone damage. For comparison purpose, damage interval has been set from .5 to .9 but lowers to 0.1 both in case (b) and (c) as shown in Fig. 9 (a) and (b) respectively.

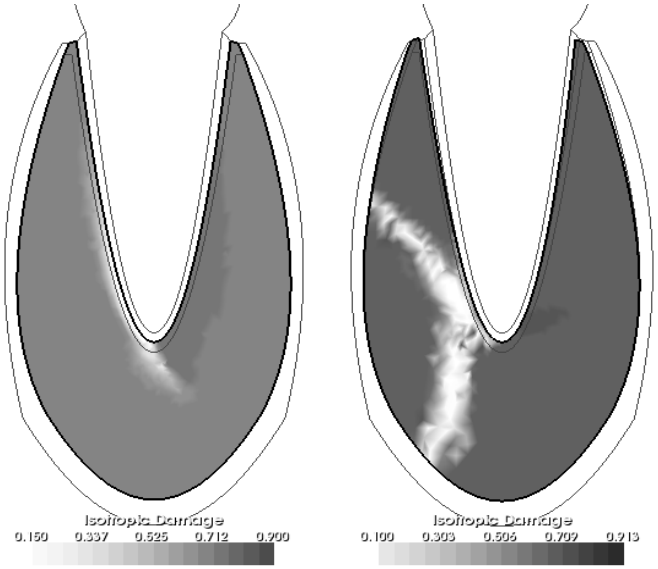
reducing $\bar{u}$ reduces U, its value stays above its homeostatic value $U^\star$ and overloaded conditions are still observed at the collar. $\dot{r}$ is therefore reduced and so is damage variation.

Concerning the stress state of the periodontal ligament, it is interesting to notice that the hydrostatic stresses are several times higher in magnitude than the shear stresses (up to 8 times for low forces). For the lower applied load, the later ones vary from 0 to $0.025MPa$ along the tooth root (highest values reached at the collar) while the hydrostatic stresses'range goes from 0 to $0.2MPa$ both in compression and traction (highest values reached at the level of the center of rotation position). The pressure is as expected the key stress in the PdL.

Qualitatively analyzing the results of this model, one can see (as in Fig. 8 and 9), as can be expected of an orthodontic treatment, that while higher loads lead to higher initial displacement, density variation of the bone can be observed only in the apex region while it is observed on the entire length of the root for smaller loads. For a given vertical position, while overloaded conditions are kept for all simulations, one can see apposition on one side (reduction of mean damage) and resorption on the other (increase of mean damage) of the root. As the remodeling law has been built so, one can see that the bone gets resorbed on the compression side and formed on the traction one along the root but also along the cortical layer when high displacements are obtained (as in Fig. 8-(c) and 9-(b)).

4. Conclusion

The present study introduces a numerical model for the simulation of orthodontic tooth movement based on the assumption that bone remodeling processes during tooth movement are controlled by elastic energy density as well as pressure state of the alveolar bone. The model is built



(a) case 1, $c_r \neq c_f$ (b) case 2, $c_r \neq c_f$

Figure 9: Alveolar bone damage - extending damage interval in Fig. 8-(b) and (c)

on a damage-repair law proposed by Doblaré and co-workers. This model has been chosen as a working framework because it is one of the few models whose stimulus variation is justified through thermodynamical concepts of continuum mechanics. It identifies the bone voids with the cavities or microcracks of other material damage models, but changes some of the standard assumptions to adapt it to the special requirements of living tissues, especially the possibility of decreasing the damage level (repair) by providing the required metabolic energy [1]. It also considers the different behavior with respect to damage criteria where damage increases in low stress regions. Its main drawback is the elastic character of the bone matrix which therefore does not include permanent strains of the matrix as a possible mechanism to get irreversible movements of a tooth in its socket.

We therefore propose an enhanced model to be used in a finite strain framework and for the specificities of alveolar bone remodeling. We assume for the bone matrix a generalized material model and considered it to be elasto-viscoplastic. The coupling of plasticity and damage is obtained using a strain equivalence approach for the effective state in a continuum damage framework. The model can therefore be used to predict long term tooth movement due both to remodeling and permanent strains of the bone matrix. The pressure dependency of alveolar bone remodeling is also treated, proposing a new remodeling rate. This model has been implemented in a in-house finite strains FE code, Metafor, as a new material law.

We also consider the potential of this pressure dependent model to predict the density evolution of alveolar bone tissue on 2D representation of a tooth submitted to orthodontic appliances. In spite of the necessary idealizations, a reliable qualitative prediction of bone density variation around the tooth is possible for porosity variations from 0% (null damage) to almost 70% (damage of 0.95), starting from an homogenized porosity of 45%.

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